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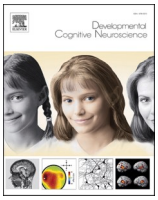

Adversity is Linked with Decreased Parent-Child Behavioral and Neural Synchrony

Caroline P. Hoyniak [a,*], Laura E. Quiñones-Camacho [a], M. Catalina Camacho [a], Jenna H. Chin [a], Elizabeth M. Williams [a], Lauren S. Wakschlag [b], Susan B. Perlman [a]

[a] Washington University in St. Louis School of Medicine, United States
[b] Northwestern University, United States

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ABSTRACT

Parent-child synchrony—parent-child interaction patterns characterized by contingent social responding, mutual responsivity, and co-regulation—has been robustly associated with adaptive child outcomes. Synchrony has been investigated in both behavioral and biological frameworks. While it has been demonstrated that adversity can influence behavioral parent-child synchrony, the neural mechanisms by which this disruption occurs are understudied. The current study examined the association between adversity, parent-child behavioral synchrony, and parent-child neural synchrony across lateral prefrontal cortical regions using functional near-infrared spectroscopy hyperscanning during a parent-child interaction task that included a mild stress induction followed by a recovery period. Participants included 115 children (ages 4-5) and their primary caregivers. Parent-child behavioral synchrony was quantified as the amount time the dyad was synchronous (e.g., reciprocal communication, coordinated behaviors) during the interaction task. Parent-child neural synchrony was examined as the hemodynamic concordance between parent and child lateral PFC activation. Adversity was examined across two, empirically-derived domains: sociodemographic risk (e.g., family income) and familial risk (e.g., household chaos). Adversity, across domains, was associated with decreased parent-child behavioral synchrony across task conditions. Sociodemographic risk was associated with decreased parent-child neural synchrony in the context of experimentally-induced stress. These findings link adversity to decreased parent-child behavioral and neural synchrony.

1. Introduction

Parent-child synchrony is defined as an observable pattern of dyadic interaction that is characterized by social reciprocity, contingent responsivity, and dyadic matching of behavior and biological rhythms. Throughout infancy and early childhood, parent-child synchrony facilitates child autonomy, self-regulatory behaviors, and social skills, and supports parent-child attachment and bond formation (Davis et al., 2017; Feldman, 2009; Harrist & Waugh, 2002; Leclère et al., 2014). Parent-child synchrony can be quantified behaviorally (e.g., specific dyadic interaction patterns), physiologically (e.g., heart rate concordance), and, more recently, using neural data (i.e., synchrony of neural activity during shared intentionality). Research suggests that behavioral, physiological, and, to a much more limited extent, neural measures of parent-child synchrony are disrupted by exposure to stressors (Azhari et al., 2019; Clearfield et al., 2014; Creaven et al., 2013; McKay et al., 1996; Nguyen et al., 2020; Suveg et al., 2016; Tarullo et al., 2017), however the mechanisms by which this occurs are not well understood. Examining the neurobiological underpinnings of observable parent-child synchrony, and how they may be disrupted by stress, will provide critical insight to how stress, broadly construed, influences parent-child outcomes.

1.1. Stress and Parent-Child Synchrony

Parent-child synchrony is thought to be context-dependent, and a growing body of research has focused on how stress, in various forms, is associated with variations in parent-child synchrony. Adversity or chronic stress includes ongoing environmental exposures such as neighborhood disadvantage, family socioeconomic status (SES), poverty, family conflict, and home chaos that are known to impact child development. Research suggests that adversity, is associated with

* Corresponding author.
E-mail address: choyniak@wustl.edu (C.P. Hoyniak).

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decreased parent-child synchrony (Azhari et al., 2019; Clearfield et al., 2014; Creaven et al., 2013; McKay et al., 1996; Nguyen et al., 2020; Suveg et al., 2016; Tarullo et al., 2017). In infancy, increased parent-child behavioral synchrony was found in dyads from higher-SES compared to lower-SES backgrounds (defined based on maternal education and social service utilization; Clearfield et al., 2014). Creaven et al. (2013) found evidence of within-dyad, dynamic concordance in heart rate, such that variations in heart rate in one member of the dyad was associated with subsequent variations in the other member of the dyad, in mother-preschooler pairs with no history of child maltreatment (i.e., abuse or neglect perpetrated by the child's mother), but not in mother-preschooler pairs with a history of child maltreatment. Higher levels of parenting-related stress have been associated with lower behavioral synchrony between parents and children ages 3 to 14 (McKay et al., 1996), and higher levels of chronic maternal physiological stress, as measured using hair cortisol, have been found to be associated with decreased parent-infant behavioral synchrony in a free play task (Tarullo et al., 2017). Taken together, these studies provide evidence suggesting that adversity impairs parent-child synchrony, which in turn could disrupt social reciprocity within the dyad.

Experimentally-induced stress in the context of dyadic synchrony research refers to stressors that are transient and induced during experimental paradigms such as challenging problem-solving tasks (such as in Lunkenheimer et al., 2017), distressing situations (such as in Pratt et al., 2015), or difficult/emotional conversations (such as in Woody et al., 2016). In Lunkenheimer et al. (2017), a structured teaching task in which parent-preschooler dyads completed difficult puzzles under time pressure led to disrupted parent-child respiratory sinus arrhythmia (RSA; an index of parasympathetic arousal) synchrony as compared to less-structured, free-play and clean up tasks. In Suveg et al. (2016) mother-preadolescent dyads showed lower levels of RSA synchrony during a child stress task (a modification of the Trier Social Stress Task; Kirschbaum et al., 1993) than during either baseline (i.e., mother-child dyads sitting side-by-side watching videos) or a discussion of topics the dyad disagrees on (e.g., chores). Although the authors hypothesize that this disruption is due to decreases in interactions between mother-child dyads during the stress task, this does not fully explain why RSA synchrony was higher during the baseline task, a low interaction context, and it is possible that the experimentally-induced stress led to a disruption in mother-child synchrony. However, Suveg et al. (2016) focused primarily on an economically-disadvantaged sample, and it is unclear how such findings would replicate in a community sample. Recent studies also demonstrate the interaction between experimentally-induced stress and psychopathology in the prediction of synchrony (Gray et al., 2017; Woody et al., 2016). In school-aged children, mother-child dyads with a history of maternal depression showed lower RSA synchrony during a negative discussion task (e.g., discussing an issue about which the dyad regularly disagrees), but not during a pleasant, vacation-planning task (Woody et al., 2016). Mother-child dyads without a history of depression showed high levels of RSA synchrony in both task conditions (Woody et al., 2016). Similarly, in a sample of preschoolers who had been exposed to at least one traumatic event, children who were diagnosed with Post-Traumatic Stress Disorder (PTSD) showed decreased parent-child RSA synchrony during a stressful memory recall task that included a discussion of the prior traumatic event (Gray et al., 2017). Preschoolers in the sample who were not diagnosed with PTSD did not show such a disruption. Although there may be reason to believe that experimentally-induced stress would directly affect parent-child synchrony, the dearth of studies that have previously explored this question highlight the need for additional research.

Also of interest is the period of time after the experimentally-induced stressor has been removed/completed, often referred to as the recovery period. The return to parent-child synchrony in the context of recovery from experimentally-induced stress may be an important indicator of adaptive parent-child functioning (Ham and Tronick, 2009) . For example, in Moore and Calkins (2004), affective synchrony in mother-infant dyads was higher in the recovery block of the still-face paradigm (i.e., the period of time post-still face) than in the normal play block (i.e., the period of time just before the onset of the still face block). These findings plausibly suggest that mothers increase the degree of coordination between their and their infant's affective states after distressing situations in order to help their child soothe and regulate after experiencing a stressor (Moore & Calkins, 2004). Previous work from our lab suggests that higher child irritability is associated with lower parent-child neural synchrony during recovery from experimentally-induced stress, suggesting recovery processes are affected by irritability/poor anger regulation (Quiñones-Camacho et al., 2019). Despite this growing literature, to our knowledge, no research has examined the interaction between adversity and experimentally-induced stress and recovery processes in the prediction of parent-child synchrony.

1.2. Adversity May Disrupt Behavioral Synchrony via the Prefrontal Cortex

Nascent work suggests that disruption of the neural circuitry that underlies behavioral synchrony (e.g., the mentalizing network) may be one mechanism through which stress has an effect on parent-child synchrony. The mentalizing network is composed of several regions throughout the frontal, parietal, and temporal cortex that coactivate during social cognition (Barrett & Satpute, 2013; Rizzolatti & Sinigaglia 2010) and undergo dramatic development during the preschool years (Deoni et al., 2015; Richardson & Saxe, 2019; Richardson et al., 2018). Specific to the reciprocity of behavioral synchrony, there is evidence that the dorsal/posterior portion of the dorsolateral prefrontal cortex (DLPFC) encodes the goal-oriented behaviors of others (Koechlin et al., 2003; Rizzolatti et al., 1988; Levy & Wagner, 2011). Successful behavioral synchrony likely requires coordinated activation of this region, which is supported by recent work examining concurrent activation of the DLPFC of two or more adults and/or children during coordinated activity (Fishburn et al., 2018; Liu et al., 2016). Two recent fNIRS studies found that higher levels of parent-reported stressors—e.g., stress about family, relationships, and finances, difficulties with parenting—were associated with decreased parent-child neural synchrony of the bilateral prefrontal cortex during a problem-solving task (Nguyen et al., 2020) and the anterior left cluster of the prefrontal cortex during a passive joint video attention task (Azhari et al., 2019). No study to our knowledge has tested whether a broader definition of adversity, including not only perceived parent stress, but also measurable socio-economic factors, impairs DLPFC activation during coordinated behavior. Elucidating this association is of critical importance for improving our understanding of the socio-emotional impairment that is associated with adversity, particularly in light of emerging evidence that an individual's neural function is sensitive to their social context (e.g., Schmälzle et al., 2017).

1.3. The Current Study

The current study examined how adversity is associated with parent-child synchrony in preschoolers, exploring how adversity interacts with a mild, transient, experimentally-induced stressor, to predict both behavioral and neural synchrony across parent-child dyads. Given a lack of consensus on how to best quantify adversity (e.g., Evans et al., 2013; McLaughlin et al., 2019; McLaughlin & Sheridan, 2016), we used a data-driven approach to measure adversity, using factor analysis to determine whether the various measures of adversity included in our study loaded onto a single factor or onto various distinct domains. The adversity indexes included in this factor analysis spanned the content areas typically included in studies of childhood adversity (e.g., economic hardship, parental psychopathology, household dysfunction and conflict; Cohen-Cline et al., 2019; Evans et al., 2013; Mersky et al., 2017),

with some exceptions (e.g., child abuse and maltreatment) due to non-inclusion of these measures at this assessment point in the overall study. We had no *a priori* hypotheses about how the different indexes of adversity included in the study would load onto domains, and instead opted for a data-driven, exploratory approach. Data were collected from children and caregivers during an interactive task designed to induce mild stress/frustration. Neural synchrony of the DLPFC was assessed using functional near infrared spectroscopy (fNIRS) hyperscanning (the simultaneous measurement of two interacting brains), and behavioral synchrony between parent-child dyads was assessed. We expected adversity to be associated with a disrupted pattern of parent-child synchrony, across both behavioral and neural domains, with dyads facing higher levels of adversity showing decreased parent-child synchrony, particularly in the context of an experimentally-induced stressor (i.e., the mild stress/frustration condition of the task). As we did not have an *a priori* hypothesis about which factors of adversity would emerge, we took an exploratory approach to examining how adversity was associated with synchrony. Additionally, given previous research suggesting sex differences in neural synchrony across both adult and child dyads (Baker et al., 2016; Reindl et al., 2018) and the possibility that the relationship between the child and the caregiver participating in the neural synchrony task (i.e., if this caregiver was a biological parent vs a caregiving grandparent) would affect overall synchrony, we also examined if these constructs were associated with parent-child synchrony in our sample and controlled for these constructs (i.e., child sex and participating caregiver status) in our analysis.

2. Method

2.1. Participants

Participants included 151 children (70 females) ages 4 – 5 years (M = 4.85 years, SD = .6 years) and their primary caregivers (93% biological mothers, 4% biological fathers, 1% adoptive mothers, 1% grandmothers, 1% not reported) who participated in a study of longitudinal development of preschool psychopathology. Children were excluded from the study if they had a neurological disorder, a history of head injury/loss of consciousness, or a diagnosed psychiatric, neurodevelopmental, or neurological disorder. In the current study, we used data acquired from the DB-DOS: BioSync task, of which 115 children (M = 4.90 years, SD = 0.61 years) had usable fNIRS data. Data loss was due to technical problems, poor sensor contact, excessive movement artifacts, and child refusal to participate. One child's data was excluded due to MRI abnormality. Note that loss of data from either the child or parent requires expulsion of data from the dyad as a whole, leading to larger rates of data loss in hyperscanning studies compared to those in which fNIRS is only collected on one individual. Additionally, one parent-child dyad that had synchrony values 4 standard deviations above the average was excluded from analysis. The racial breakdown of the final sample of 115 children (52 females) was 69% White, 21% Black/African American, 1% Asian, and 10% Bi- or Multi-racial, with 5% of primary caregivers reporting that their child was of Hispanic or Latino descent. Primary caregivers in the final sample were primarily college educated (13% High School degree or less; 26% some college/Associate's degree; 26% Bachelor's degree; 38% Master's degree or higher) and married (77%; 5% separated/divorced; 5% no contact; 5% co-parenting; 4% other). Families reported a range of incomes (15% less than $20,000; 16% $20,000 – $39,000; 13% $40,000 – $59,000; 23% $60,000 – $99,000; 33% over $100,000). All study procedures were approved by the Institutional Review Board at the University of Pittsburgh.

2.2. Measures

2.2.1. Adversity

We opted to take a data-driven approach to quantifying adversity, using factor analysis to combine across plausible adversity indexes and identify domains of adversity. Several indexes were examined for possible inclusion in our measure of adversity (described below). Descriptive statistics for, and correlations between, each variable considered for the adversity index are included in Table 1.

2.2.1.1. Area Deprivation Index. The Area Deprivation Index (ADI; University of Wisconsin School of Medicine Public Health, 2015) is a composite measure that provides an index of relative sociodemographic disadvantage for a given Census Block Group. ADIs use information gathered from the 2015 American Community Survey, combining across 17 variables: education distribution, median family income, income disparity, occupational composition, unemployment rate, family poverty rate, percentage of population below 150% of poverty rate, single-parent households, home ownership, median home values, median monthly mortgage payments, median rent, household crowding, percentage of households without access to a phone, plumbing, or a motor vehicle, English language proficiency, percentage urban population, percentage immigrant population (Singh, 2003). ADI values range from 1 to 100, reflecting the percentile ranking of the given Census Block Group in comparison with the rest of the nation. ADI's were computed for each child based on primary caregiver-reported home addresses. Higher scores were indicative of higher levels of disadvantage. The ADI has been demonstrated to have adequate psychometric properties (Singh, 2003) and has been shown to be associated with a variety of negative health outcomes (Kind & Buckingham, 2018).

2.2.1.2. Family Income. Primary caregivers reported on the family's combined yearly household income prior to taxes on an 8-point scale ranging from 1 (*less than $20,000*) to 8 (*more than $250,000)*. This variable was reverse-scored, so that higher scores reflected lower incomes.

2.2.1.3. Social Service Utilization. Primary caregivers reported on their use of social services during the past year, including their use of Medicaid, the Special Supplemental Nutrition Program for Woman, Infants, and Children (WIC), food stamps, welfare, aid for dependent children, and disability compensation. Participants received a score of 1 for each social service they reported using, and these scores were summed to create an index of total social service utilization. The Cronbach's alpha value for the index of social service utilization was 0.71.

2.2.1.4. Single Parent Status. Primary caregivers reported on the child's caregiving situation. From this, we identified children who only had one caregiver involved in their life (biological or otherwise) and had no contact with another caregiver. This variable was scored such that children with single caregivers were given a score of one, while all other children were given a score of 0.

2.2.1.5. Caregiver Education Level. Primary caregivers reported on his or her education level (i.e., highest degree earned), as well as the education level of the child's other biological parent (if applicable). Education levels were rated on a 7-point scale, from 1 (*No degree or diploma)* to 7 (*Doctorate, Ph.D., M.D., J.D., other)*. This variable was then reverse-scored, so that higher scores reflected lower education levels.

2.2.1.6. Maternal Age at Child Birth. Primary caregivers reported on the child's birth date as well as the child's biological mother's birth date. From this we derived the child's biological mother's age at child birth (M_{age} = 30.54 years, SD_{age} = 5.31 years). Biological mothers who were 19 years old or younger when the child was born, suggesting that they were "teenaged" (4 mothers in the final sample), were given a score of one. All other mothers were given a score of 0. We opted to dichotomize this variable to reflect the heightened level of psychosocial risk that teenage mothers experience in comparison with non-teenaged mothers (Mollborn, 2017).

Table 1
Descriptive statistics and bivariate correlations between adversity indexes.

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Area Deprivation Index	1								
2. Family Income	-.72**	1							
3. Social Service Utilization	.64**	-.77**	1						
4. Single Parent Status	.39**	-.45**	0.39**	1					
5. Caregiver Education Level	-.65**	.73**	-.69**	-.49**	1				
6. Maternal Age at Childbirth	.17*	-.20*	.21**	.12	-.20*	1			
7. Household Chaos	.16^	-.09	.15^	.08	-.13	.00	1		
8. Family Conflict	.16^	-.23**	.16^	.41**	-.20*	.16*	.45**	1	
9. Primary Caregiver Psychopathology	.28**	-.29**	.30**	.28**	−0.27**	.14^	.45**	.52**	1
N	114	115	115	111	112	115	115	115	115
M	55.99	4.13	1.17	0.13	4.72	0.04	14.29	2.47	53.24
SD	29.72	2.18	1.45	0.33	1.18	0.2	3.69	2.18	34.36

Note: ^ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$ – all two-tailed.

2.2.1.7. Household Chaos. Household chaos was examined using primary caregiver-reports on an abbreviated form of the Confusion, Hubbub, and Order Scale (CHAOS; Matheny et al., 1995) . Households high on the construct of home chaos are those characterized by a sense of confusion, rush, and disorganization, and typically lack a sense of order or a consistent routine (Matheny et al., 1995). Our abbreviated version of the CHAOS scale included 6 items for which primary caregivers indicate whether a given item is true for their home, either 0 (*No*) or 1 (*Yes*). Scores on each item were summed to create an overall index of home chaos, with higher scores indicating more chaotic homes. The Cronbach's alpha value for the home chaos index was 0.54, reflecting the abbreviated nature of the version of the CHAOS scale we administered.

2.2.1.8. Family Conflict. Family conflict was examined using the Family Environment Scale (FES; Moos & Moos, 1994), a widely-used measure of family climate. We only administered the conflict and cohesion subscales of the FES (due to these being the aspects of family process of greatest interest in the wider study) but focus the current analysis only on the conflict subscale, because it is a more direct measure of overt conflict within the family structure. Primary caregivers indicated whether each item on the conflict subscale (e.g.., "Family members often criticize each other") was true about their family, either 0 (*No*) or 1 (*Yes*). Scores on each subscale item were summed to create an overall index of family conflict, with higher scores indicating higher levels of conflict. The Cronbach's alpha value for the family conflict subscale was 0.78, in line with the internal consistency data for the conflict subscale reported by Boyd et al. (1997).

2.2.1.9. Primary Caregiver Psychopathology. Primary caregiver psychopathology was examined using primary caregiver reports on the Adult Self-Report Inventory – 4 (ASRI; Gadow et al., 2004). The ASRI, a reliable and valid self-report measure of psychopathology, includes 135 items that assess symptoms across the major categories of psychopathology, including anxiety disorders, mood disorders, eating disorders, somatoform disorders, psychotic disorders, child-onset disorders (i.e., Attention Deficit Hyperactivity Disorder, Oppositional Defiant Disorder, Conduct Disorder), substance-use disorders, and Borderline Personality Disorder. Primary caregivers indicated whether a given symptom describes their overall behavior on a 4-point scale, from 0 (*Never)* to 3 (*Always)*. The ASRI was scored using the symptom severity scoring method: the individual's scores on the items corresponding to each disorder were summed to create an overall symptom severity score for each diagnostic category. Category scores were summed to create an overall index of parent psychopathology, with higher scores indicating increased parent psychopathology. The Cronbach's alpha value for the overall index of parent psychopathology was 0.97.

2.2.1.10. Adversity Factor Analysis. To dimensionally reduce and combine the indexes of environmental adversity into theoretically relevant factors, we used a minimum residual factor analysis with an oblique rotation. Factor analysis was conducted in R (R Core Team, 2019) using the psych package (Revelle, 2018). Prior to factor analysis, all adversity indexes, except for the dichotomized indexes (i.e., single parent status and maternal age at childbirth), were converted to proportions of maximum. The number of factors retained during PCA was determined using a Parallel Test (Horn, 1965), in which a scree plot of the data was plotted against the scree plot of normally-distributed, random data. Factor retention was determined based on how many factors represent a meaningful signal (i.e., the point at which the data scree plot is equivalent to the random scree plot). The Parallel Test identified that a solution with two factors would be the most appropriate for the data. One-, two-, and three- factor solutions were all fitted, and the factor solution with the best fit, the two-factor solution, was chosen. Findings suggested that this two-factor solution accounted for 57% of the variance and demonstrated adequate fit statistics (RMSEA = 0.09; see Table 2). Factor 1 accounted for 38% of the variance, and indexed sociodemographic risk including the following variables: 1) Area Deprivation index, 2) family income, 3) social service utilization, 4) single parent status, and 5) caregiver education level. Standardized loadings ranged from 0.44 - 0.91. Factor 2 accounted for 19% of the variance and indexed familial risk including the following variables: 1) household chaos, 2) family conflict, and 3) primary caregiver psychopathology. One variable, maternal age at child birth (dichotomized to identify teenaged mothers), did not load onto either factor and was excluded from further analysis. Factor scores were extracted based on the two-factor solution using regression-based weights and used in further analysis.

2.2.2. Laboratory-induced stress during parent-child interaction

Neural synchrony was assessed using the DB-DOS: BioSync task (Quiñones-Camacho et al., 2019), a modification of the Disruptive

Table 2
Factor loadings and communalities from factor analysis with oblimin rotation for adversity indexes.

	Factor 1: "Sociodemographic Risk"	Factor 2: "Familial Risk"
Area Deprivation Index	.78	
Family Income	.91	
Social Service Utilization	.83	
Single Parent Status	.44	
Caregiver Education Level	.83	
Household Chaos		.60
Family Conflict		.80
Primary Caregiver Psychopathology		.67
Factor 1 and 2 correlation: $r = .35$		

Note: Loadings less then .3 are omitted, which included loadings on both factors for maternal age at childbirth.

Behavior Diagnostic Observation Schedule (DB-DOS) a developmentally sensitive, observational index of child disruptive behavior (Wakschlag et al., 2008). The DB-DOS: BioSync contained two experimental conditions, one in which frustration was induced for both members of the dyad, and one in which the dyad was given time to recover from the frustration-induction condition. During the DB-DOS: BioSync, primary caregivers and children were seated at a table, and asked to participate in two tasks: a puzzle completion task (i.e., frustration) and a free-play task (i.e., recovery). During the puzzle completion task, which always occurred first and was designed to elicit mild stress/frustration, the dyads were told that they would need to complete "a lot" of tangram puzzles in order to win a prize. They were instructed to work as a team, as quickly as possible, while following instructions presented on a computer screen in the room. The puzzle portion of this task was divided into 4 blocks, each lasting 2 minutes with 5 different possible puzzles to solve during each block, and 15-second inter-block intervals. Dyads were instructed to complete as many of the 5 puzzle choices during the 2-minute window as possible, and while solving, the computer screen displayed a countdown clock indicating how much time was remaining in the block. Frustration/stress was induced by 1) using tangram puzzles that were, unbeknownst to the dyad, too difficult for the child's developmental level, 2) by cutting the time to solve short (1:45 minutes instead of the promised 2:00 minutes), and 3) by having attractive toys present in the room, but not allowing the child to play with them. The reward component of the task (i.e., winning a prize for completion) was included for the purposes of increasing parent and child buy-in for the task, and it allowed us to induce frustration by blocking that reward for a period of time (i.e., delaying the receipt of the reward).

During the free-play task, which was designed to be a recovery period, the dyads were given access to the attractive toys in the room. Dyads were allowed to play with the attractive toys for 10 minutes. To mirror the structure of the puzzle completion task, the free-play task consisted of 4 blocks of 2 minutes followed by a 15-second inter-block interval, with a new toy added each block.

2.3. Parent-Child Behavioral Synchrony

An in-house coding scheme was used to quantify parent–child behavioral synchrony during both conditions of the DB-DOS: Biosync task (i.e., frustration and recovery; Quiñones-Camacho et al., 2019). Synchrony was defined as reciprocal, coordinated engagement demonstrated through shared attention, shared topic, and contingent responding. Each second of the parent-child interaction task was coded as either synchronous (e.g., reciprocal communication, eye contact, coordinated behaviors) or asynchronous (e.g., no turn taking, communication, or mutual engagement for more than three seconds). For a dyad to be coded as synchronous, they needed to exchange three verbal or behavioral turns (given that reciprocal interactions are necessary to establish synchrony). Dyads were coded as synchronous until there was a break in reciprocal exchanges (i.e., more than three seconds passed since the dyad had showed reciprocal responding). Parent-child behavioral synchrony was calculated as the amount time (in seconds) the dyad was synchronous during each condition (i.e., frustration and recovery) of the task. Of the original 151 participants, 127 cases were codable (missingness due to problems with the video camera and audio of the interaction), and 20% of these codable cases were double-coded for reliability (Kappa = .81). Of these 127 cases, 98 also had useable fNIRS data.

2.4. fNIRS Data Collection

A NIRScout fNIRS system (NIRx Medical Technologies LLC, Glen Head, NY) was used to collect continuous-wave fNIRS data from both the parent and the child during the DB-DOS: Biosync task. Eight LED light sources emitted light at 760 and 850 nms, which were detected by 4 photodiode light detectors, with ten measurement channels per wavelength. Optical signals were collected at 15.625 Hz. Sensors were placed on a neoprene head cap using a source-to-detector distance ranging from 2.9 to 3.1 cm. Head caps were positioned according to the international 10-20 coordinate system, with detectors over AF7/AF8 and F7/F8 and sources over FC5/FC6, F5/F6, AF3/AF4, and Fp1/Fp2. Channels therefore covered the middle frontal gyrus (MFG), the inferior frontal gyrus (IFG), the superior frontal sulcus, and the inferior frontal sulcus of each hemisphere of the PFC (see Fig. 1). Each subject was registered to the Colin27 Brain Atlas (Holmes et al., 1998). Hair located under each optode was manually parted to improve signal detection. A calibration sequence was conducted for each dyad before data acquisition. Optodes were manually adjusted to ensure adequate signal quality before data collection.

2.5. fNIRS Preprocessing

Preprocessing and subsequent activation analysis were conducted in MATLAB (MathWorks, Natick MA USA) using the NIRS Brain AnalyzIR toolbox (Santosa et al., 2018). Raw intensity signals were first converted to changes in optical density. To correct for motion artifacts arising from excessive head movements, we used the Temporal Derivative Distribution Repair (TDDR) technique (Fishburn et al., 2019). TDDR uses a robust regression approach to remove large fluctuations in the optical density signal (attributed to motion artifacts), while retaining smaller fluctuations (attributed to hemodynamic activity). Corrected optical density signals were then downsampled to 4 Hz, and slow wave drifts in the signal were removed using a high-pass filter of 0.01 Hz. Optical density signals were then transformed into oxygenated hemoglobin concentrations using the modified Beer-Lambert Law.

2.6. Parent-Child Neural Synchrony

Neural synchrony was defined as the association between concurrent lateral PFC activation of the parent and the child, calculated separately for the frustration condition and the recovery condition of the DB-DOS: BioSync. To quantify neural synchrony, timings were standardized across all participants. Signals were then whitened to remove temporal autocorrelations, a common source of noise that may inflate correlation estimates, using an autoregressive model (Santosa et al., 2017). Robust correlation coefficients were then calculated between participants using a robust regression approach, in which the geometric mean is calculated for the robust regression coefficients obtained from regressing channel X onto channel Y and vice-versa. Synchronization, across all possible channel pairs, was quantified using Fisher r-to-z transforms of absolute values of correlation coefficients. Reciprocal connections were enforced in order to reduce the number of unique connections.

The p-value associated with each synchrony value was computed using a permutation test, in which the proportion of values from null-pairings (e.g., random, non-paired dyads) that were equal to or greater than the observed value was determined. Adjusted z-values were then derived from these estimated p-values using the inverse cumulative density function for the standard normal distribution. These values were examined using a mixed effects model, with task condition included as a fixed effect and dyad modeled as a random effect. The presence of synchrony was assessed for each condition by applying the t-contrast corresponding to a 1-sample t-test. Corresponding p-values were corrected for multiple comparisons using the Benjamini-Hochberg False Discovery Rate correction (Benjamini & Hochberg, 1995; corrected p-values represented as a q-value) across all unique channel pairs. The mean of the adjusted z-values was computed across significant ($q < .05$) channel-pairs for each dyad. This process reduces data for each dyad to a single measurement, collapsing across significant channels. These adjusted z-values were used as the quantification of mean parent-child neural synchrony in subsequent analysis.

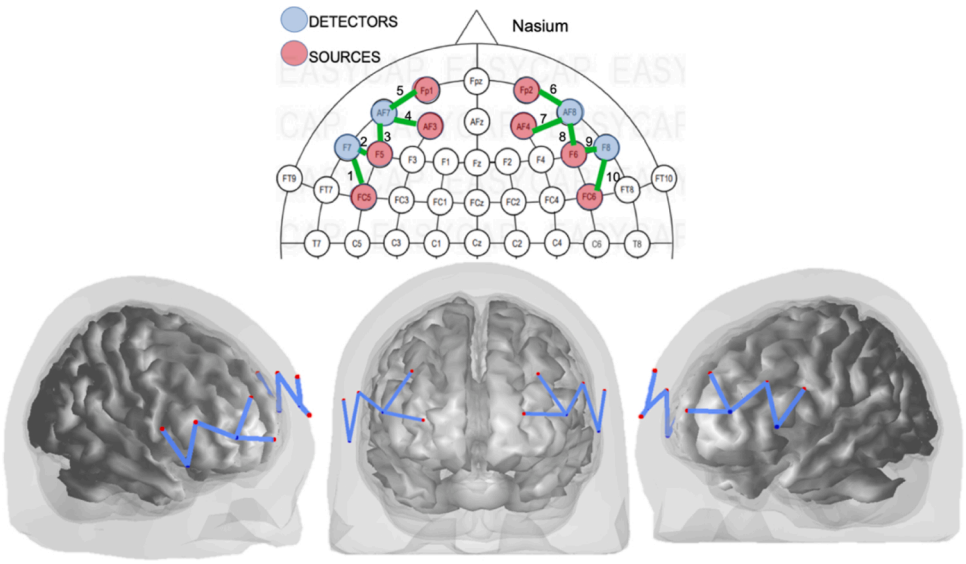

Fig. 1. Channel and optode arrangement for each member of the dyad during the DB-DOS:BioSync.

2.7. Analysis Plan

To explore the association between adversity and synchrony, we examined the association between the two dimensions of adversity that emerged during the factor analysis (factor 1— sociodemographic risk and factor 2 – familial risk) and parent-child behavioral and neural synchrony using Pearson correlations. We examined the association between adversity and synchrony separately for the two conditions of the DB-DOS: BioSync, frustration and recovery. Next, significant associations were further probed using multiple regression to control for child sex and participating caregiver relationship to child (i.e., biological mother vs. other caregiver). All analyses were conducted in R (R Core Team, 2019).

3. Results

3.1. Parent-Child Behavioral and Neural Synchrony

A full description of parent-child behavioral and neural synchrony results is presented in Quiñones-Camacho et al. (2019). Briefly, significant parent-child neural synchrony was found for both task conditions, and there was a wide distribution of parent-child neural synchrony across dyads (Frustration condition: $M = 0.44$, $SD = 0.48$, range = -0.71 – 1.56; Recovery condition: $M = 0.44$, $SD = 0.47$, range = -0.72 – 1.96). There were no significant differences in either behavioral or neural synchrony across task conditions ($t[96] = 1.10$, $p = .28$ and $t[114] = 0.10$, $p = .92$, respectively). Behavioral synchrony was associated with neural synchrony in the frustration condition of the parent-child interaction task ($r = .21$, $p = .04$), but not in the recovery condition ($r = -.04$, $p = .73$). Differences on key demographic variables between low- and high-synchronous dyads (determined using a median split) for both behavioral and neural synchrony across task conditions are presented in Supplemental Table S1.

3.2. Association Between Adversity and Parent-Child Behavioral Synchrony

We examined the association between factor scores across each adversity dimension (i.e., sociodemographic and familial risk) and behavioral synchrony in both the frustration and recovery conditions of the parent-child interaction task using Pearson correlations (see Table 3). A significant association between behavioral synchrony during the frustration condition and both adversity dimensions emerged, such that children experiencing higher levels of sociodemographic and familial risk showed lower parent-child behavioral synchrony during the frustration condition (see Fig. 2A and B). Additionally, a significant association emerged between behavioral synchrony in the recovery condition and familial risk, such that children experiencing higher levels of familial risk (but not sociodemographic risk) showed lower parent-child behavioral synchrony during the recovery condition (see Fig. 2C).

Table 3
Descriptive statistics and bivariate correlations between adversity indexes.

		Factor 1 – Sociodemographic Risk	Factor 2 – Familial Risk	Mean (SD)
Behavioral Synchrony	Frustration	-.36**	-.27**	292.22 (128.39)
	Recovery	-.19^	-.26*	305.77 (106.25)
Neural Synchrony	Frustration	-.35**	-.14	0.44 (0.48)
	Recovery	-.02	-.08	0.44 (0.47)

Note: ^ $p \leq .10$, * $p \leq .05$, ** $p \leq .01$ – all two-tailed.

3.3. Association Between Adversity and Parent-Child Neural Synchrony

We also examined the association between factor scores across each adversity dimension and neural synchrony in both the frustration and recovery conditions of the parent-child interaction task using Pearson correlations (see Table 3). We found a significant association between sociodemographic risk (factor 1) and neural synchrony during the frustration condition, such that increased exposure to sociodemographic risk was associated with lower levels of parent-child neural synchrony during the frustration condition of the task (see Fig. 2D). There was no significant association between sociodemographic risk and parent-child neural synchrony in the recovery condition, and there was no significant association between familial risk (factor 2) and parent-child neural synchrony during either the frustration or recovery conditions.

3.4. Multiple Regression Analysis with Control Variables

Next, we re-examined the significant correlations between the adversity dimensions and both behavioral and neural synchrony, controlling for child sex and participating caregiver's status as a biological

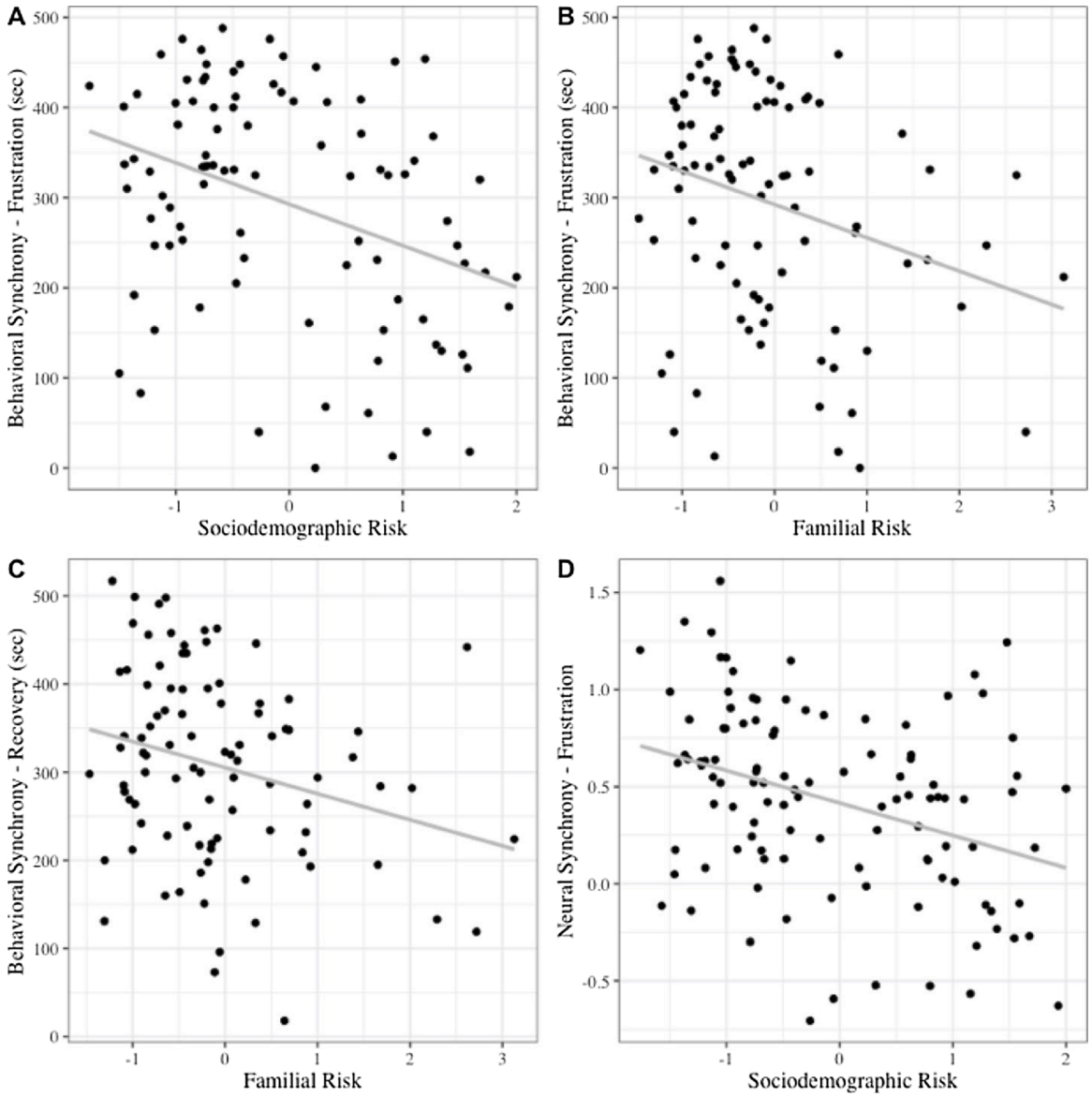

Fig. 2. Significant correlations between adversity factors and behavioral and neural synchrony.

mother (Table 4). All examined associations remained significant even after accounting for the plausible covariates of child sex and participating caregiver's relationship to child.

4. Discussion

The current study examined parent-child synchrony in the context of adversity and experimentally-induced stress in a sample of preschoolers. Behavioral and neural synchrony were examined in the context of a mild, experimentally-induced stressor and a recovery condition. Child adversity was examined across two, empirically-derived domains: 1) sociodemographic risk and 2) familial risk. Findings suggested broad associations between adversity and reduced parent-child behavioral synchrony in the context of experimentally-induced stress. The findings with neural synchrony were less robust, and only partially supported our hypotheses: sociodemographic risk was associated with decreased parent-child neural synchrony in the context of frustration induction, but not during subsequent recovery. Additionally, familial risk was not associated with parent-child neural synchrony in either condition.

Given the many possible ways of classifying and quantifying adversity, we opted to use a data-driven, factor analytic approach to grouping the measures of adversity included in the current study. The two domains of adversity identified, which we termed sociodemographic risk and familial risk, are similar to those identified in prior research, including Gubhaju et al. (2013) which identified two higher-order factors of adversity, material disadvantage (which included family composition, social service utilization, financial hardships, etc.) and psychosocial disadvantage (which included parent-parent relationships, parent well-being, etc.). Separate lines of work have also identified dimensional structures of adversity, including identifying higher-order factors of threat (e.g., experiences of abuse or trauma) and deprivation (e.g. poor cognitive stimulation; Sheridan & McLaughlin, 2014), as well as, child maltreatment and household dysfunction (Mersky et al., 2017). However, these lines of research focus on aspects of adversity (e.g., child maltreatment) that are not well represented in our study, and so we were unable to examine whether similar dimensions emerged in our data.

Adversity, across domains, was found to be significantly associated with lower parent-child behavioral synchrony across task conditions. The only exception was the association between sociodemographic risk and parent-child behavioral synchrony in the recovery condition, which did not reach traditional levels of significance, but was trending. These findings correspond with previous research suggesting that stress/

Table 4
Multiple regression analyses controlling for plausible co-variates.

	β	*SE*	*p*-value
Behavioral Synchrony Frustration			
Factor 1: Sociodemographic Risk	-.37	12.96	<.001
Child Sex	.08	26.29	.44
Participating Caregiver Relationship to Child	.03	63.73	.77
	$F(3,87) = 4.55$,	$p = .005$,	$R^2 = .14$
Behavioral Synchrony Frustration			
Factor 2: Familial Risk	-.27	14.04	.01
Child Sex	.01	27.03	.92
Participating Caregiver Relationship to Child	-.01	65.25	.93
	$F(3,87) = 2.36$,	$p = .07$,	$R^2 = .08$
Behavioral Synchrony Recovery			
Factor 2: Familial Risk	-.35	11.73	.02
Child Sex	.13	22.58	.21
Participating Caregiver Relationship to Child	.03	54.51	.80
	$F(3,87) = 2.68$,	$p = .05$,	$R^2 = .08$
Neural Synchrony Frustration			
Factor 1: Sociodemographic Risk	-.33	.04	<.001
Child Sex	-.13	.09	.16
Participating Caregiver Relationship to Child	-.02	.18	.80
	$F(3,104) = 5.6$,	$p = .001$,	$R^2 = .14$

Note: Child Sex: 1=male, 2=female; Participating Caregiver Relationship to Child: 1 = Biological Mother, 0=Other Caregiver.

adversity, in its various forms, may have a disruptive effect on parent-child behavioral synchrony (Clearfield et al., 2014; Creaven et al., 2013; McKay et al., 1996; Tarullo et al., 2017). Perhaps unsurprisingly, parent-child behavioral synchrony showed a more robust association with adversity than parent-child neural synchrony. This may reflect a weak concordance between behavioral and neural synchrony in some contexts or behavioral synchrony being more closely tied to neural regions which were not measured by the specific region-of-interest fNIRS method employed here.

The novel contribution of the current study is our focus on parent-child neural synchrony in the context of adversity and experimentally-induced stress. Our neural synchrony findings suggest that certain parent-child dyads, namely those experiencing higher levels of sociodemographic risk, are more susceptible to the effects of experimentally-induced frustration on neural synchrony. In the face of this frustration, these dyads displayed lower levels of neural synchrony, which has been associated with lower levels of shared attention, engagement, mutual responsivity, and poorer problem-solving capacities (Nguyen et al., 2020: Quiñones-Camacho et al., 2019). It is possible that, for dyads facing adversity, the cognitive resources reserved for coping with mild, transient stressors are more easily disrupted, leading to less synchronous interactions. Previous research suggests that exposure to higher levels of adversity is associated with altered physiological responses to experimentally-induced stress in both adults (Gump & Matthews, 1999; Steptoe et al., 2002) and youth (Evans et al., 2007).

Although prior meta-analytic work suggests that recovery mechanisms after experimentally-induced stress are also disrupted in individuals facing higher levels of general life stress (Chida & Hamer, 2008), our findings suggest that, in families facing higher levels of sociodemographic risk, the removal of the mild, transient stressor enabled the dyad to return to more adaptive levels of neural synchrony during the recovery condition. There are several plausible explanations for this finding. First, prefrontal neural synchrony may still be flexible in parent-preschooler dyads. The prefrontal cortex undergoes dramatic development during the preschool period (Deoni et al., 2015; Tsujimoto, 2008), thus it is possible that the consequences of adversity on prefrontal cortex functioning (for review, see Pechtel & Pizzagalli, 2011; Sheridan & McLaughlin, 2014) have not yet been fully realized in preschoolers. A second, complementary explanation is that the dyads experiencing higher levels of sociodemographic risk were employing adaptive regulation strategies that allowed them to overcome disruptions in synchrony after the removal of the experimentally-induced stressor. This is consistent with previous work demonstrating increased activation of limbic and subcortical regions during socioemotional cognition in individuals who have experienced high levels of adversity (Callaghan & Tottenham, 2016; Kim et al., 2013). In other words, there is evidence that bottom-up processes—which are connected to autonomic functioning—impair the top-down cognition necessary to engage in deliberate behavioral reciprocity (Lieberman, 2007). Thus, the preschool period may represent a period of relative plasticity in the mentalizing network. Additionally, the recovery condition of the task may reflect ongoing relationship reparation processes induced by the removal of the experimentally-induced stressor. In the context of these reparative processes, synchrony may be increased even in the context of early adversity. Without a baseline condition in which to examine neural synchrony prior to the onset of the experimentally-induced stressor, however, this is a speculative interpretation. Future research examining neural synchrony in the context of experimentally-induced frustration should include a true neutral baseline condition occurring before the frustration condition. Adding this baseline will help to distinguish between neural synchrony occurring as a result of recovery and repair processes and typical patterns of neural synchrony for the dyads in the absence of frustration or any other negative emotions. With a baseline condition, the nature of the dynamic changes in neural synchrony that occur in the context of both experimentally-induced stressors and adversity can be more fully explored. Additionally, this would enable the computation of a change or difference score that more directly quantifies resting-state to frustration-state changes in neural synchrony. As the current study lacks the ability to compute a resting-to-frustration difference score in activation, this must be kept in mind when interpreting our findings.

Existing research on the effect of stress and poverty on neural functioning may help to inform interpretations of the mechanisms underlying our findings. First, it is important to note that the regions we measured spanned across emotion regulation and executive control networks, and it is possible that disruptions in either or both of these networks contribute to the effects observed in the present study. Second, we did not examine activation. Instead, we examined co-activation, the degree to which the child and parent activate and deactivate the LPFC at the same time. Given the spatial limitations of fNIRS, we were limited to the LPFC and do not know if activations or deactivations of the LPFC relative to other brain regions underlie the coactivation patterns observed during the task. For example, previous work suggests that poverty is associated with decreased activation of the LPFC during emotion processing and regulation (Kim et al., 2013; Liberzon et al., 2015). It is therefore possible that poverty is associated with alterations in emotion regulation which may affect co-regulation in the DB-DOS: BioSync task. Further, there is extensive evidence to suggest differences in executive control networks in children and adults who have experienced poverty or early life stress (see Johnson et al., 2016 and Palacios-Barrios and Hanson, 2018 for review). Since early stress is associated with alterations in neuronal development (McLaughlin et al., 2009) and there is extensive white and gray matter PFC development occurring during the preschool years (Lebel & Deoni, 2018), it is possible that contextual risk is associated with LPFC coactivation via alterations in the neuronal structure and function of this region. Future research should seek to carefully delineate which mechanisms underlie these differences and examine if they are uniform across the emotion regulation and/or executive control networks.

As we used a data-driven, factor analytic approach to identify the various domains of adversity, we did not have *a priori* expectations about what adversity categories would be identified. However, given that

multiple, distinct domains were identified, this allowed us to examine the differential associations between these domains and synchrony. Although behavioral synchrony was associated with both domains of adversity (i.e., sociodemographic and familial), only sociodemographic risk was associated with parent-child neural synchrony. The index of sociodemographic risk contains specific indicators that reflect economic hardship (e.g., low family income, reliance on social services for needed food and medical assistance, residing in a more economically deprived area, etc.). An important strength of our factor analytic approach is that we reduced shared variance across variables by combining a number of variables into a single measure. However, it is important to keep in mind that each of these indicators might have unique effects on parent-child synchrony (e.g., a single parent might develop a more adaptive, synchronous relationship with their child due to increased time spent together or might have undue caregiving and earning burdens that reduces their capacity to spend one-on-one time with their child, or some combination of both of these processes). As such, future research with larger samples designed to disentangle associations with individual measures will be an important next step in the literature.

Although synchrony has been shown to be affected by other factors associated with household dysfunction, such as parenting stress (McKay et al., 1996; Azhari et al., 2019) and maternal depression (Feldman, 2007), our findings did not replicate these prior findings. Our index of familial risk, which included measures of household chaos, family conflict, and parent psychopathology and may serve as a plausible index of household dysfunction, was not associated with neural synchrony. The risk factors we classified as "familial risk" (e.g., home chaos, family conflict) might be considered to be one mechanism through which more distal stressors such as neighborhood disadvantage or lower income-to-needs ratios affect parent-child relationships. As such, it might be expected that we would find a larger association between synchrony and familial risk than between synchrony and sociodemographic risk, given the more proximal nature of this domain of stressors. However, this was not the case, and only sociodemographic risk was associated with neural synchrony. There are several plausible explanations for our lack of findings. First, our index of familial risk was derived from only one developmental time point and relied entirely on primary caregiver-reports of home functioning. A more comprehensive index, including information from multiple time points to establish the chronic nature of exposure to chaotic, conflictual households (which may be a pre-requisite for a transient stressor to show effects on neural development; McLaughlin et al., 2019) would enable us to better explore the unfolding association between chronic, familial risk and parent-child neural synchrony. Similarly, as we relied on parent-reports of home environments, it is possible that parents demonstrating lower synchronous behaviors are poorer reporters of their home environments, as such, future studies should consider using more direct, first hand reports of the family environment. It is, of course, plausible that there is truly no association between familial risk and neural synchrony, but future studies that include a longitudinal design and direct observations of home environment, will be better able to clarify the nature of this association.

Additionally, although prior research has examined how psychopathology, in both parents and children, interacts with experimentally-induced stress to affect parent-child synchrony (Gray et al., 2017; Woody et al., 2016), we included our index of parental psychopathology in our measure of adversity for several reasons. First, empirically, parent psychopathology loaded with factor 2, familial risk, in our factor analysis, and theoretically, this is consistent with conceptualizations of parent psychopathology as a risk factor for more adverse early environments. Next, prior research examining the effect of parent psychopathology on synchrony in the context of experimentally-induced stress has primarily focused on clinical populations (e.g., mothers with depression; Woody et al., 2016). As our sample was a community sample, we did not have a sufficient number of parents with psychopathology to focus on specific clinical groups.

A strength of the current study is that we used an empirical, data-driven approach to conceptualize adversity across the domains of sociodemographic and familial risk. This allowed us to explore the specific aspects of child adversity that were associated with parent-child synchrony (Farah, 2017; McLaughlin & Sheridan, 2016; McLaughlin et al., 2019). Our index of sociodemographic risk included a number of factors associated with SES, creating a more robust measure than the traditional reliance on only family income or only parent education/occupation (Farah, 2017). Additionally, we also explored how adversity interacts with experimentally-induced stress to predict parent-child neural synchrony, an experimental paradigm that allowed us to further probe neural synchrony in the context of various forms of stress. An additional strength of the current study is that we demonstrate that the regulation of frustration at the dyadic level is not a stable trait but is context-dependent. While previous research has often focused on context in terms of with *whom* the child is interacting (e.g., a stranger or a parent) and the quality of the child's general relationship with caregivers, the current study focuses on context in terms of wider family background in the form of adversity. These findings suggest that the presence of sociodemographic risk affects observed dyadic frustration regulation. This is the first study, to our knowledge, to demonstrate this at both the neural and dyadic levels.

There are limitations worth noting in the current study. As mentioned, our relatively limited capacity to quantify household dysfunction (i.e., relying only on primary caregiver reports at a single time point) could have affected our capacity to find an association between familial risk and neural synchrony. Additionally, while fNIRS imaging provides a more relaxed and reliable neuroimaging experience for young children than EEG or MRI approaches, fNIRS imaging is limited to coarse recordings of the cortical surface.We were not able to examine whether subcortical regions of the brain that are implicated in stress reactivity (e.g. the amygdala or hippocampus) or brain regions, beyond prefrontal regions, in the wider mentalizing network are differentially synchronous within the dyad. Finally, in the current study, neural synchrony was quantified using a single synchrony value that represents the magnitude of synchrony throughout a task condition. Although this is consistent with the technique utilized by other researchers using hyperscanning to quantify neural synchrony (Miller et al., 2019; Nguyen et al., 2020; Reindl et al., 2018), as this area of research continues to develop, we should find ways to more explicitly model within-condition and within-task changes in neural synchrony, as this is a dynamic construct even within a single interaction context.

5. Conclusions

The current study examined how parent-child behavioral and neural synchrony is associated with adversity and experimentally-induced stress. Parent-child behavioral synchrony was broadly associated with adversity, across task conditions. Neural synchrony, however, was only associated with sociodemographic risk, such that sociodemographic risk was associated with decreased parent-child neural synchrony in the context of experimentally-induced frustration, but not during a subsequent recovery period. These findings improve our understanding of how experimentally-induced stress interacts with adversity to affect parent-child synchrony, and may have important implications for understanding how day-to-day, relatively transient frustrations (e.g., mild child noncompliance or running late) have an effect on parent-child synchronous interactions in families facing adversity. These families may be more susceptible to interactional dysregulation in the face of daily stressors, but appear to recover at rates similar to families facing lower levels of sociodemographic risk. These findings may have implications for intervention efforts aimed at improving parent-child relationships and interactions, such as Parent-Child Interaction Therapy and Parent Management Training. Including a focus on improving a parent's capacity to manage day-to-day frustrations, possibly through the use of cognitive or behavioral strategies, could be especially

beneficial for improving parent-child interactions in families facing high levels of sociodemographic risk.

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Declaration of Competing Interest

The authors report no declarations of interest.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.dcn.2021.100937.

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